

AMENDMENTS TO THE CLAIMS

1. (Currently Amended) A method of treating a patient having an ischemic disease, a dental disease, smoker's leg, a diabetic ulcer, or stimulation of wound healing in said patient in need thereof, said method comprising administering Use of a nucleic acid encoding TBK-1 or a functional active derivative thereof. for the preparation of a pharmaceutical composition for the treatment of ischemic or dental diseases, smoker's leg and diabetic ulcers or for the stimulation of wound healing.

2. (Currently Amended) The method ~~[[use]]~~ of claim 1, wherein the nucleic acid induces the production of VEGF.

3. (Currently Amended) The method ~~[[use]]~~ according to any of claims 1 or 2, wherein the nucleic acid induces the formation of vascular vessels.

4. (Currently Amended) A method for the diagnosis of ischemic disease, a dental disease, smoker's leg, a diabetic ulcer, a wound healing disorder, cancer, hyperplasia, tumor progression, rheumatoid arthritis, psoriasis, arteriosclerosis, retinopathy, osteoarthritis, endometriosis or chronic inflammation in a patient, said method comprising detecting the level of Use of

- a) TBK-1,
- b) a functional active derivative thereof, and/or
- c) a nucleic acid encoding TBK-1, ~~and/or~~

said method comprising the use of a means for the detection of the molecules of sections a), b), or c).

- ~~d) means for the detection of the molecules of sections a), b), c) or d)~~

~~for the preparation of a diagnostic agent for the diagnosis of ischemic or dental diseases, smoker's leg and diabetic ulcers, wound healing disorders, cancer, hyperplasia, tumor progression, rheumatoid arthritis, psoriasis, arteriosclerosis, retinopathy, osteoarthritis, endometriosis or chronic inflammation.~~

5. (Currently Amended) A method for inhibiting or reducing angiogenesis in the treatment of cancer, hyperplasia, rheumatoid arthritis, psoriasis, arteriosclerosis, retinopathy, osteoarthritis, endometriosis or chronic inflammation in a patient in need thereof, said method comprising administering a TBK-1 inhibitor. ~~Use of a TBK-1 inhibitor for the preparation of a pharmaceutical composition for inhibiting or reducing angiogenesis in the treatment of cancer, hyperplasia, rheumatoid arthritis, psoriasis, arteriosclerosis, retinopathy, osteoarthritis, endometriosis or chronic inflammation.~~

6. (Currently Amended) The method ~~[[use]]~~ of claim 5, wherein the inhibitor inhibits the production of VEGF.

7. (Currently Amended) The method ~~[[use]]~~ of any of claims 5 or 6, wherein the inhibitor inhibits the formation of vascular vessels.

8. (Currently Amended) The method ~~[[use]]~~ of any of claims 5 or 7, wherein the inhibitor is selected from the group consisting of antisense oligonucleotides, antisense RNA, siRNA, aptamers and Low molecular weight molecules (LMWs).

9. (Currently Amended) The method ~~[[use]]~~ of claim 8, wherein the LMWs bind to the ATP-binding site of the kinase domain of TBK-1.

10. (Currently Amended) The method [[use]] of any of claims 4 to 9, wherein the disease is cancer, preferably selected from the group consisting of brain cancer, pancreas carcinoma, stomach cancer, colon carcinoma, skin cancer, especially melanoma, bone cancer, kidney carcinoma, liver cancer, lung carcinoma, ovary cancer, mamma carcinoma, uterus carcinoma, prostate cancer and testis carcinoma.

11. (Original) A method for the identification of an anti-cancer drug, wherein

- a) a potential TBK-1 interactor is brought into contact with TBK-1 or a functional derivative thereof, and
- b) binding of the potential interactor to TBK-1 or the functional derivative thereof is determined, and
- c) the anti-angiogenic capacity of the potential interactor is determined.

12. (Original) The method of claim 11, wherein the anti-angiogenic capacity is determined by measuring the inhibition of VEGF production.

13. (Original) The method of any of claims 11 or 12, wherein the potential interactor is provided in the form of a chemical compound library.

14. (Original) The method of claim 13, wherein the chemical compound library consists of a group of molecules or substances that bind to the ATP binding site of the kinase domain of TBK-1.

15. (Original) The method of any of claims 11 or 14, wherein the method is carried out on an array.